road which has no sign to direct us forward to our desired destination. There are no crossroads in the learned professions. There is only one road—a straight road, well marked and centuries old.

On the gravestone which marks the resting place of a great American lawyer and statesman, William Henry Seward, are these words, "He was faithful."

Let us be faithful to our professional heritage. Let us dedicate our individual efforts in the future so that when the time comes for us to give way to our younger brothers who will follow in our footsteps it may be said of each of us, "He was faithful."

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VISUAL DISTURBANCES FOLLOWING HEAD INJURY*

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OF all the special senses, vision and hearing are valued far above the others. The mechanisms concerned in normal vision are numerous and, in the majority of serious head injuries, some part of this elaborate scheme is temporarily or permanently affected (Russell [25] and Strauss and Savitsky [27]). In this paper, injuries of the optic nerve and their posterior connections are considered from the neurosurgical viewpoint.

A few generalities may first be made regarding head injuries. It is demonstrated frequently that an insignificant blow on the head may cause extensive intracranial damage of almost any variety. The fact that consciousness was not lost is no assurance whatever that the brain or cranial nerves have escaped injury. A patient may have a large depressed fracture with contused brain beneath, and not even be dazed by the impact. This is illustrated in another way by Mock,[19] who, in 1939, estimated that in the United States every year there were 150,000 fractured skulls and 450,000 serious cerebral injuries. The presence of a linear fracture of the skull is of little consequence if other intracranial injury has not been received. A basal fracture tends to radiate along the bone that is most easily broken or separated. Finally, in civil life, the majority of head injuries that affect vision are inflicted by falling in one way or another (Davidson [8]).

The portions of the optic pathways most frequently injured are the optic nerves, the optic chiasm, the visual cortex, and the visual association areas (Cohen [5]).

INJURIES OF THE OPTIC NERVE

Excluding bullet wounds of the head, the orbital portion of the optic nerve is almost never injured by bone fragments (Ballantyne,[2] Pringle [21]). This is largely true even of the portion in the optic canal. Most orbital fractures are in the thin roof, being a continuation back of a fracture following a blow on the forehead or face. Callan [4] theorized that such a fracture tended to follow the frontoethmoidal suture to its union with the frontosphenoidal and ethmosphenoidal sutures, where it continued back across the lesser wing of the sphenoid to involve the optic canal. Pringle expressed the contrary view that the fracture line more commonly traveled medially in the ethmosphenoidal suture, sparing the optic foramen. He examined at autopsy 136 cases of fractured skull and no direct injury of the optic nerve was found. In thirty-eight cases of orbital roof fracture on one or both sides, twelve of them had a fracture into one or both optic foramina. He found anterior clinoids fractured in five instances, and the posterior clinoids fractured in six. Naffziger [20] observed a fractured anterior clinoid process that had compressed the optic nerve to about one-third of its normal width. Barkan and Barkan [3] pointed out that the optic nerve in the optic canal is attached by arachnoid to the dural sheath more intimately superomedially than elsewhere. They felt that a fracture of the upper inner wall of the canal would be particularly likely to damage the nerve by splitting the arachnoid. Rollet, Paufique, and Levy [24] felt that x-rays could be expected to reveal optic canal fracture if taken soon after the accident. Davidson,[8] in twenty-two cases of optic canal fracture, found x-ray evidence of narrowing in only one.

Monocular blindness, following a head injury, is usually attributed to hemorrhage into the dural sheath of the optic nerve, especially if the blindness comes on or if it increases after the accident. In some instances the hemorrhage is continuous with a subdural collection, but several cases have been reported in which separate intravaginal bleeding was found. Cohen and Miceli [18] reported cases in which the subarachnoid space around the optic nerve was distended by clear fluid that compressed the nerve severely. The typical history of intravaginal hemorrhage is of a diffuse blow on the forehead, with or without symptoms of concussion, after which vision in one eye is impaired or absent. Barkan and Barkan found sector-field defects accompanying optic nerve injuries. They postulated that, when the macular bundle is spared, the pupil reacts to light, but that if the macular bundle be injured there is a central scotoma and absence of the direct pupillary response to light, even though the eye be not completely blind. They stated that a sector defect up to and including the macula was characteristic of an injured optic nerve. Various investigators have found that, if blindness persist, optic atrophy appears in a few weeks and sometimes as early as six days (Davidson). In a few cases of blindness from slight optic nerve injury, spontaneous recovery has occurred in from a few hours to several months.

Some authors (Pringle, Eagleton [9]) have advocated, and employed, surgical exposure of the optic nerve to drain an intravaginal hemorrhage. The results have not been good, but usually the interval between accident and operation was very

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long. Possibly earlier operation might have been more successful. Eagleton described an operative approach to the orbital roof by dividing the upper anterior portion of the temporal muscle and entering the anterior fossa beneath the outer part of the frontal lobe.

Three cases are on record of fracture in the optic foramen without appreciable effect on the optic nerve until some months later, when callus formation compressed the nerve, producing monocular disturbances (Lillie and Adson,[16] Clovis Vincent [28]).

Adhesive arachnoiditis (Ballantyne, Craig,[17] Holmes,[13] Cushing,[7] Russell [26]) is a poorly explained condition that may affect the intracranial portion of the optic nerve, causing signs and symptoms suggestive of pressure by a tumor. The history is sometimes obtained of a head injury a few months before, with the later onset of gradually dimming vision. Operation reveals thickened arachnoid around the optic nerves and liberation of the adhesions sometimes clears up the condition or halts its progress.

REPORT OF CASE

R. S., twenty years old, was involved on September 13, 1939, in a car accident, being thrown to the road. He was unconscious momentarily, and then dazed for a short while. He had a contusion with macerated scalp in the right temple, fracture of the right zygoma, fracture of the nose and a fracture through the left petrous bone. Both eyes were swollen shut. Three days later, when the bandages were removed, the right eye was blind and the left face paralyzed. A month after the accident the right eye was still blind, and the disc was bluish-white and sharply outlined. There was well-developed papilledema in the left eye. Seven months after the accident the right disc was yellowish-white and the left still showed papilledema. The left visual field, on the perimeter, was contracted to 50 degrees with a blind spot twice normal size. Sixteen months after the accident the right eye was still blind and the left disc blurred, but less elevated. He complained of slightly impaired vision on the left and found that the eye often felt dry while he was driving in his car. It seems likely that, whatever happened to the right optic nerve, the left was compressed by hemorrhage in the dural sheath. X-rays of the optic canals showed normal contour. The dryness was explained by interruption of the secretory fibers to the lacrimal gland as they traversed the left middle ear, in the greater superficial petrosal nerve.

INJURY OF THE OPTIC CHIASM

The chiasm may be torn when a severe fracture splits the skull in the olfactory groove. Coppez [6] decided from experiments that a separation of bone in the midline of 26 millimeters or more was likely to tear the chiasm. The tear is most often in the midline, from in front, resulting in a bitemporal hemianopia. Sometimes, one optic nerve is completely divided as well, so that there is vision only in the other nasal field. Rand [22] found in two cases a chiasmal lesion after a blow on the vertex. He explained it by a downward thrust of the cranial contents with a sudden forcing forward of the fluid in the third ventricle, against the chiasm.

INJURY OF THE VISUAL CORTEX

It is in this location that many head injuries have their effect on vision. The calcarine cortex may be the seat of concussion, edema, contusion, laceration, compression by depressed skull fragments or compression by a hematoma. The plan of cortical localization, as described by Holmes,[14] has been found to agree with clinical observations of other authors. The upper and lower lips of the calcarine sulcus represent the corresponding upper and lower halves of the retina. Peripheral vision is received, finally, in the anterior portion of the calcarine area, whereas macular vision is represented in the region of the occipital pole. Holmes further assigned the horizontal meridian of the visual field to the lips of the calcarine sulcus, and the vertical meridian to the cortex in the depths of the sulcus.

Careful neurologic examination is necessary to locate the intracranial lesion, but, to decide its pathologic character, one must rely largely on the course of the condition. In mild concussion of the occipital lobes, when consciousness is not lost, there may be partial or total blindness in one or both visual fields for a few minutes or even an hour or two (Anderton [1]). If localized cerebral edema develop, the visual complaints may not be at their worst at once, and will last for, perhaps, a few days. Hine [12] stressed the fact that after a concussion a slight lesion may only produce a visual field defect for colored objects. This dissociation of white and colored objects was not found in cases of intracranial hematoma with field defects. He pointed out that when a blow is received in the occipital region, it is usually above the occiput, so that the upper calcarine area is the more severely affected, resulting in an inferior quadrant hemianopia. He reviewed six cases of occipital concussion with field defects and most of them had a split macula. Strauss and Savitsky also stated that the field defect might be for colored objects only. They found ring scotomas after head injury, detecting them only on the tangent screen. No good explanation was offered to account for these scotomas, but it was suggested that fatigue might constitute one factor. Ingham and Lyster [15] reported a patient with depressed fracture above the occiput who did not lose consciousness. He saw a flash of light and was then momentarily blind, following which a symmetrical horizontal inferior hemianopia remained. Two days later the hemianopia was present only for smaller test objects.

In cerebral contusion the symptoms are more severe and of still longer duration. In addition, there is subarachnoid hemorrhage, so that a spinal puncture would show bloody fluid and there might be photophobia from meningeal irritation. H. W. Grant [11] found fields concentrically contracted to 10 degrees in a man who had been unconscious for about ten minutes after a car accident. In two months there was complete recovery. Rand reported concentrically contracted fields in several patients after head injury, but attributed it to hysteria. It is likely that a contusion, and certain that a laceration, would leave some permanently damaged brain tissue.

Riddoch [23] stated that, in recovering lesions affecting the visual fields, appreciation of motion was the earliest function to appear, and that if this were found in the first few months after injury

the prognosis was good for continued return of vision.

INJURY OF VISUAL ASSOCIATION AREAS

This is the cortex adjacent to the calcarine area, including most of the occipital lobe and particularly its medial surface. Here are located the higher mental processes concerned with vision, the essential function being to interpret what is seen and to integrate the information in the light of previous experience. Disturbances of this function may cause such symptoms as obscure difficulty in reading (Edwards [10]) and in following the thread of a story, difficulty in understanding what is read, or in recognizing what is seen. In right-handed individuals the speech center resides in the left hemisphere, which is then called the major hemisphere. Injuries of the visual association areas in the major hemisphere are more likely to interfere with visual-speech mechanisms than if the injuries occur in the minor hemisphere. Ballantyne reported two cases of word-blindness after a head injury.

Holmes concluded that lesions on the lateral surface of the hemisphere, even as far forward as the posterior parietal region, may disturb the higher visual perceptual functions, resulting in loss of visual orientation and localization in space, loss of perception of depth and distance, also visual agnosia, and loss of visual attention. Strauss and Savitsky describe many subtle forms of similar disturbance. One patient could read without being able to recognize the individual letters. Westcott [29] examined seventy-two patients with so-called accommodative asthenopia after head injury, but decided that, in 65 per cent of them, the real trouble was in the higher cerebral association centers.

The primary objective in examining the visual mechanism after a head injury is to decide on any treatment which may improve the final outcome. The second aim is to form an opinion regarding the prognosis. The appropriate treatment is indicated only by an accurate diagnosis as to location of the lesion and its pathologic characteristics. Surgical measures sometimes can relieve symptoms due to compression by bone, edema, hemorrhage or by adhesions. The difficult part of the work is early diagnosis, so that any indicated measure can be applied as soon as possible after the condition appears. The risk of such surgical intervention is usually not great and is negligible when balanced against the possibility of obtaining better vision for the patient.

SUMMARY

1. In civil life most injuries producing monocular disturbances are the result, not of damage by bone fragments, but of compression by other means, particularly hemorrhage in the dural sheath of the nerve. The prognosis is grave, but it is conceivable that if such a hemorrage were drained promptly vision might recover better.

2. Bony callus may form at a fracture line in the optic foramen, causing compression of the optic nerve.

3. Adhesive arachnoiditis may develop some months after a head injury and cause serious impairment of vision in both eyes.

4. Injury of the visual cortex produces a wide variety of signs and symptoms, sometimes with dissociation for color, appreciation of movement, and for form.

5. Many patients with vague but persistent difficulty in reading, following a head injury, have a cortical lesion in the visual association areas. Formerly the tendency was to attribute the complaints to a neurosis if no eye muscle imbalance could be found.

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ASPHYXIA NEONATORUM *

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WHEN discussing the resuscitation of the newborn infant with asphyxia, it has been customary to begin with a consideration of what factors normally operate to initiate respiration. However, it is now generally accepted that the respiratory mechanism does not begin its function at birth, but that it normally begins some time during fetal life *in utero*. This has been ably demonstrated experimentally in various animals by Wislocki,[1] by Snyder and Rosenfeld,[2] by Bonar,[3] and by Windle and Becker.[4] There can be little doubt that this occurs in the human fetus, as it has been observed directly during cesarean operations, and indirectly through the abdominal wall of the mother.[5,6] The suggestion has even been made that the resultant aspiration of amniotic fluid may normally aid the "dilatation and differentiation of the alveoli."[6]

Furthermore, it has been shown that these fetal respiratory movements, in animals, are sensitive to drugs and gases administered to the mother. Maternal anoxemia of a degree which only stimulates maternal respirations may depress or abolish the fetal respiratory movements; CO_2 deficit, likewise, will depress fetal respirations; but CO_2 excess has little, if any, stimulating effect. Drugs which barely produce maternal analgesia may completely abolish the fetal respiratory movements.[2,4]

Therefore, it would seem more constructive that we should forsake the old concept that respiration starts at birth. It would be better to follow the suggestion of Snyder, Bonar and others, and consider an infant's first respiratory effort after birth not as the initiation of a new mechanism, nor as the awakening of a dormant function, but as the continuation of a vital process which functioned from time to time *in utero* and which was interrupted during birth.

It is not difficult to understand why the respiratory mechanism is so often seriously altered by delivery. A newborn infant may well be considered as suffering from some degree of shock as the result of even minimal birth trauma.[7] In addition, the baby is often under the influence of depressant drugs administered to the mother. Maternal analgesics are seldom without some effect on the infant; often this effect is considerable, as in the case of morphin, ether, sodium pentobarbital, or paraldehyd.[8] And, finally, in addition to the two handicaps—shock and narcosis—there is often the possibility of other less obvious factors, such as hemorrhage or defects (organic or functional) in one or more vital organs.

When, as a result of these factors, shock and narcosis, respiratory function fails to recover quickly, another seriously damaging process, anoxemia, begins to add further injury. The effect of anoxemia on the vital nerve centers, which control respiratory and vasomotor reactions, is to depress further their sensitivity. Yandell Henderson[9] has contended that, inasmuch as the respiratory center is depressed by oxygen lack, it requires a stronger stimulus to produce activity. However, there is little direct evidence to support this contention, and there is experimental data tending to refute it. Schmidt[10] has shown experimentally that when anoxemia has progressed to the point where the respiratory center is depressed, stimulants such as CO_2, various drugs (atropin, camphor, strychnin, caffein), or increased acidity, not only fail to stimulate but even act as further depressants.

Eastman[11] has measured the principal biochemical changes which occur in the blood of asphyxiated newborns and compared them with similar studies of normal newborns. In asphyxiated infants there is primarily a decrease in the oxygen content of the blood, a marked increase in lactic acid, a lowered blood p^H, and an increased CO_2 tension. In some of Eastman's patients the blood p^H fell to the lowest limits compatible with life, and in two fatal cases the p^H was below 7.0. Eastman feels that this severe acidosis is due to fixation of base by accumulated lactic acid, and to a CO_2 excess which cannot be removed through the normal channels of placenta or lung. Similar findings have been reported by Wilson, Torrey, and Johnson.[12]

In view of these considerations, supported by clinical experiences, it would seem that the fundamental immediate need of the asphyxiated baby is oxygen, and that respiratory stimulants such as CO_2 or various drugs are superfluous and may be even harmful.

SEQUELAE OF NEONATAL ASPHYXIA

Realizing that anoxemia, if severe or prolonged, causes cell death, the question, naturally, arises whether prenatal or neonatal asphyxia of a degree insufficient to cause the death of the total organism may result in permanent defects of the nervous system. We are interested especially in injury to the nervous system because nerve tissue is especially sensitive to anoxemia and an injured nerve cell cannot be replaced; whereas injuries to other organs can, to a much greater extent, be functionally repaired.

There are experimental data concerning the effects of anoxemia on the nerve cells of animals. Anoxemia will raise the threshold and decrease the conductivity of isolated nerve segments.[13] Yant and his coworkers[14] found that in dogs anoxemia

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† Since this paper was written, an excellent report of the pathological effects of asphyxia has been published by Clifford. (Clifford, S. H., Journal of Pediatrics, 18:567, 1941.) This study was made on eleven infants asphyxiated in utero and delivered by cesarean section, thus eliminating possible effects of birth trauma.